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Stobadine inhibits lysosomal enzyme release in vivo **and in** vitro

Navarova J, Macickova T, Horakova K, Urbancikova M

LIFE SCIENCES
65 (18-19): 1905-1907 OCT 1 1999

Document type: Article **Language:** English **Cited References:** 13 **Times Cited:** 1

Abstract:
This study investigated the ability of stobadine, an effective cardioprotective drug with antiarrhythmic, antihypoxic and oxygen free radical scavenging properties, to protect cells against cyclophosphamide-induced toxic and cytotoxic damage in **vivo** and in **vitro**. Cyclophosphamide-induced toxic damage in female ICR mice was accompanied by marked increase in the activity of lysosomal enzymes in the spleen and kidney. Administration of stobadine prior to cyclophosphamide inhibited these biochemical changes. The in **vivo** protective effect of stobadine was comparable with its in **vitro** effect established in **HeLa cells**.

Author Keywords:
stobadine, lysosomal enzymes, in **vivo** stobadine effect, in **vitro** stobadine effect

KeyWords Plus:
CYCLOPHOSPHAMIDE, METABOLISM, TOXICITY, MICE

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Publisher:
PERGAMON-ELSEVIER SCIENCE LTD, OXFORD

IDS Number:
245GX

ISSN:
0024-3205

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Comparison of isoproterenol - Induced changes in lysosomal enzyme activity in vivo **and in** vitro

Macickova T, Navarova J, Urbancikova M, Horakova K

GENERAL PHYSIOLOGY AND BIOPHYSICS
18: 86-91 Sp. Iss. SI OCT 1999

Document type: Article **Language:** English **Cited References:** 16 **Times Cited:** 1

Abstract:
Isoproterenol was used as a drug which, when administered in high doses, is able to induce lysosomal enzyme activity changes in in **vivo** conditions. We correlated lysosomal enzyme activity in the absence and presence of isoproterenol, obtained in whole animals and in HeLa and HeDG2 cells in tissue culture. In **vivo** experiments: male Wistar rats (270 300 g) were treated subcutaneously with isoproterenol in various doses. Effect of isoproterenol on lysosomal enzyme activity was assayed in the heart after differential centrifugation. In **vitro** experiments: Isoproterenol in concentrations 0.1-100 mu g/ml was added to HeLa and HepG2 cells and the activity of lysosomal enzyme was measured in the cell homogenate. In the sedimentable and nonsedimentable fractions of the rat my ocardium, the isoproterenol-induced changes in the activity of lysosomal enzyme were time-and dose-dependent. In **HeLa cells**. isoproterenol administration caused a dose-dependent increase of lysosomal enzyme activity, while in HepG2 cells the activity remained unchanged. Thus the isoproterenol-induced changes in lysosomal enzyme activity in the rat myocardium were comparable with the results found in **vitro** in **HeLa cells**.

Author Keywords:
isoproterenol, lysosomal enzyme activity, rat, in **vivo**, in **vitro**

KeyWords Plus:
INDUCED MYOCARDIAL-INFARCTION, ISCHEMIA, RATS, NECROSIS, CELLS

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Publisher:
GENERAL PHYSIOL AND BIOPHYSICS, BRATISLAVA

IDS Number:
297XX

ISSN:
0231-5882

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AATEX 1, 2-9, November 1990

IN VITRO CYTOTOXICITY TEST USING RABBIT CONJUNCTIVA, RABBIT CORNEA AND HELA CELLS AS ALTERNATIVES FOR THE DRAIZE EYE IRRITATION TEST

TADASHI OKUBO[1], KEIKO HIRAIWA[1], SHIGEMI KINOSHITA[1] AND MASAMI WATANABE[2]

[1]Research Laboratory, POLA Corporation, 27-1 Takashimadai Kanagawa-ku Yokohama 221, Japan; [2]Division of Radiation Biology, School of Medicine, Yokohama City University, 3-9 Fukuura Kanazawa-ku Yokohama 236, Japan.

SUMMARY

In vitro cytotoxicities of 7 detergents, 5 shampoos and 3 rinses were determined on the basis of the colony forming abilities of three types of cells, primary rabbit conjunctival (RCN) cells, corneal (RC) cells and established HeLa cells. We compared the cytotoxicities of each cell type *in vitro* and the Draize eye irritation test *in vivo,* and compared the cytotoxicities among three types of cells. There was a good correlation between the cytotoxicities of each ceils *in vitro* and the Draize score *in vivo,* and a correlation among three types of cells. The same sensitivities among the three types of cells were observed. These data suggest that, using either RCN, RC or HeLa cells, the cytotoxicity test *in vitro* may be useful as a substitute for the Draize eye irritation test.

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MAP4 is the in vivo **substrate for CDC2 kinase in** HeLa cells**: Identification of an M-phase specific and a cell cycle-independent phosphorylation site in MAP4**

Ookata K, Hisanaga S, Sugita M, Okuyama A, Murofushi H, Kitazawa H, Chari S, Bulinski JC, Kishimoto T

BIOCHEMISTRY
36 (50): 15873-15883 DEC 16 1997

Document type: Article **Language:** English **Cited References:** 72 **Times Cited:** 24

Abstract:
We reported previously that cdc2 kinase decreased the microtubule-stabilizing ability of a major HeLa cell microtubule-associated protein, MAP4, by phosphorylation in **vitro** [Ookata, K., et al. (1995) J. Cell Biol. 128, 849-862]. An important question raised by this study is whether MAP4 is indeed phosphorylated by cdc2 kinase at mitosis in **vivo**. We present here evidence that cdc2 kinase is the major M-phase MAP4 kinase, and, further, we identify two phosphorylation sites within the proline-rich domain of MAP4. Metabolic P-32 labeling showed the increased phosphorylation of MAP4 at mitosis. A specific inhibitor of cdc2 kinase, butyrolactone I, inhibited phosphorylation of MAP4 both in mitotic **HeLa cells** and in the mitotic HeLa cell extract. The phosphopeptide map analysis revealed the high similarity of in **vivo** labeled mitotic MAP4 to that phosphorylated by cdc2 kinase in **vitro**. Ser-696 and Ser-787, both of which lie within SPXK consensus sequences for cdc2 kinase, were identified as phosphorylation sites in the proline-rich region of MAP4 in **vivo** and in **vitro**. Immunoblotting with antibodies that recognize the phosphorylation state of Ser-696 or Ser-787 showed that Ser-787 in the SPSK sequence was specifically phosphorylated at mitosis while Ser-696 in the SPEK sequence was phosphorylated both at mitosis and in interphase. These results suggest that cdc2 kinase directly regulates microtubule dynamics at mitosis through phosphorylation of MAP4 at a number of sites, including Ser-787.

KeyWords Plus:
MICROTUBULE-ASSOCIATED PROTEINS, XENOPUS EGG EXTRACTS, DYNAMIC INSTABILITY, TAU-PROTEIN, MOLECULAR CHARACTERIZATION, DEPENDENT KINASES, STARFISH OOCYTES, BINDING, MITOSIS, INVITRO

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Publisher:
AMER CHEMICAL SOC, WASHINGTON

IDS Number:
YN267

ISSN:
0006-2960

Molecular Mechanisms of Metal Toxicity and Carcinogenicity
Environmental Health Perspectives 102, Supplement 3, September 1994

[Citation in PubMed] [Related Articles]

Protective Effects of Thiol Compounds on Chromate-induced Toxicity *In Vitro* and *In Vivo*

Nobuyuki Susa, Shunji Ueno, and Yoshinori Furukawa

School of Veterinary Medicine and Animal Sciences, Kitasato University, Twada, Aomori, Japan

Abstract

The effects of thiol compounds (l-cysteine ethyl ester, 2,3-dimercaptosuccinic acid, or 2,3-dimercapto-1-propanesulfonic acid) on the toxicity induced by chromate (potassium dichromate) were investigated in HeLa cells and mice. Chromate-induced cytotoxicity evaluated by inhibition of cell growth and chromium content of the cells was diminished by all of the thiol compounds tested when the cells were incubated in the medium with both chromate and one of the thiol compounds. In mice injected ip with a thiol compound immediately after injection of chromate, mortality, ornithine carbamyl transferase activity in the serum, and chromium content in the liver were diminished remarkably compared with mice injected with chromate alone. These thiol compounds also caused an increase of urinary chromium excretion. These results suggest that the thiol compounds tested are useful for treating chromate-induced toxicity when they are given immediately after intake of the metal. -- **Environ Health Perspect** 102(Suppl 3):247-250 (1994).

Key words: chromate, l-cysteine ethyl ester, 2,3-dimercaptosuccinic acid, 2,3-dimercapto-1-propanesulfonic acid, HeLa cells, mice, cytotoxicity, thiol compounds

This paper was presented at the Second International Meeting on Molecular Mechanisms of Metal Toxicity and Carcinogenicity held 10-17 January 1993 in Madonna di Campiglio, Italy.

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Last Update: January 6, 1999